GENERAL DISCUSSION: SESSION II CARBON MONOXIDE

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DR. MERRIL EISENBUD: I asked earlier this morning how much was being spent on automobile emission controls and nobody seemed to know, but I do not think that anybody will argue with the assumption that it is several billion dollars a year. As I said earlier, the figures that I have used range from 11 to 20 billion dollars per year.

The point is that this is by far the largest investment that this country or any country has ever made in the name of public health. The whole health budget of the New York City Health Department is about $100 million a year, and here we spend somewhere between 11 and 20 billion dollars a year eliminating an air pollutant on which Dr. Coburn says no research is being done. I would quite agree with that; I think the level of research is minuscule but I think that this is a blindness that we have throughout the environmental field. We seem to be quite willing to spend billions of dollars to control pollutants on the basis of a rather flimsy research base, but an investigation to establish whether an adequate basis exists cannot get hundreds of thousands or a few million. I hope that as this conference goes on, some of the gaps in our knowledge will be identified.

MRS. VANDY BRADOW (Environmental Protection Agency): You stated that the investment to control automotive pollutants is the largest single investment for public health. I do not have the numbers at my fingertips, but I do believe that the investment for control of sulfur oxides from stationary sources is probably quite a lot larger.

DR. EISENBUD: I believe the figure for sulfur oxides is $4 billion a year. This one seems to be pretty well established.

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MRS. BRADOW: Are you discussing stationary or mobile sources?

DR. EISENBUD: Stationary sources. These are Council on Environmental Quality figures on sulfur dioxide.

DR. BERNARD GOLDSTEIN (Rutgers Medical School): Dr. Coburn, I definitely agree with your commercial in favor of more research in this area. Obviously, it is criminal that this is not being done. But what do you do as a physician with patients who have arteriosclerotic heart disease, who have angina? Do you try to alter the way they are exposed to automobile exhaust, how they handle themselves in traffic? Do you try to keep pregnant women away from high traffic areas because of the considerations you have given us?

DR. RONALD COBURN: I am a chest physician. I tell emphysema patients to stop smoking.

DR. GOLDSTEIN: What do you advise your cardiologist friends?

DR. COBURN: Well, I do not know that I have any. I do have one, yes, but he has never asked me for advice. I suppose that if I had angina pectoris myself I would stay out of high carbon monoxide concentrations. By high I mean more than about 20 parts per million over a short period of time. I would not take a job where there was high carbon monoxide industrial exposure.

DR. MAURICE SHILS (New York Academy of Medicine): What are the relative contributions of automobile and stationary sources of pollution? If one takes carbon monoxide as an example, what proportion comes from automobiles? Dr. Eisenbud indicated that although there had been a decrease, there are areas of New York City where the level of carbon monoxide is in the range which, according to your statistics, you would consider potentially hazardous for pregnant women and for people with angina in terms of oxygen transfer. Where is the CO coming from?

DR. COBURN: I wish Dr. Ferrand were here to help me with this. Basically, in most big cities it depends on how much industry is there and so forth, but the automobile is the source of at least 80% of carbon monoxide. That is true in New York, at least in 1970. I suppose that there are point sources as well, home heating and some that have been pointed out today, but in terms of the totals, which is the concern I have, it looks like the automobile is the big thing.

DR. EISENBUD: There are really two aspects to the answer. One is the total quantity and the other is the concentration at which it is released. Actually, the largest sources of carbon monoxide by far are the natural

sources, but they are very diffuse. Carbon monoxide is produced by decaying organic matter, and it is produced very substantially by the flora of the oceans. Automobiles, of course, are far less diffuse, but far more concentrated than the automobile is the cigarette. Although the cigarette may not have an important effect on general air unless one is in a football stadium, as you pointed out, in a badly ventilated room with many smokers the person is exposed to approximately 40,000 parts per million, I believe, in the mainstream of the cigarette. This is by far the most concentrated source, and, in terms of the numbers of grams of carboxyhemoglobin produced in a city like New York, it is probably the largest.

DR. COBURN: I do not think so, compared with all the automobile sources. The cigarette source is greatly diluted, also. One is not exposed to 40,000 parts. One's alveolar CO concentration may be perhaps 100 parts per million right after one inhales. This is diluted tremendously.

DR. EISENBUD: Yes, I understand. I was talking about the mainstream.

MR. MICHAEL JONES (U.S. Environmental Protection Agency): With regard to allocation of funds for health research on automotive-related pollutants, I think that you are quite right that there has not been a lot of research on the health effects of carbon monoxide over the last few years. However, the total amount spent for the control program does include the hydrocarbon oxidant or NOx problem. There has been considerable effort in that particular research area. In addition, we recognize the gaps in the carbon monoxide health research area, and we are taking steps right now to get additional funding in 1981 and 1982, both in clinical and in epidemiologic studies, to assist in that data base.

DR. ARTHUR STERN (University of North Carolina): I have noted that the draft Environmental Protection Agency air quality criteria document for carbon monoxide shows that the critical values used are exposures of angina patients. These critical studies put angina patients on either bicycle ergometers or on treadmills and exercised them to the point that they were forced to stop by anginal pain.

As an angina patient myself, I would not want the entire population of the United States to have their automobiles controlled because I could not control the onset of my angina by a little bit of self-restraint and by the use of the nitroglycerin tablets commonly carried by angina sufferers. To require everybody in the United States to be limited in their purchases of automobiles and other equipment to protect the few of us who can protect ourselves is in my opinion a rather poor policy.

DR. COBURN: I think the reasoning is that once one shows an adverse effect in a susceptible population, and it has been shown in several susceptible populations, it may project to millions of people. One demonstrates a toxic effect that may project to many more than the full number of angina pectoris people. Adverse effects have been shown in normal people exercising and in angina people. A body of literature about other susceptible populations, including the fetus, and a number of psychological studies, while not as solid, suggest adverse effects at these low levels. These are worrisome studies, and those responsible for protecting these people are conservative people. If future research shows that only patients with cardiovascular disease and angina are susceptible to 3% blood carboxyhemoglobin levels and that nobody else is susceptible, then I would think that the standards should rise.

DR. STEVEN HORVATH (University of California): One thing that people frequently forget with regard to the CO problem is that nobody is exposed to nine parts per million. Actually, we are exposed at times to around 100 or 150 parts per million. The bolus effect is probably more important than the steady state effect. In California, if one drives on any of the freeways such as the Long Beach Freeway, one may be exposed to 50 parts per million without any trouble. In fact, that is what Aronow did with his patients; he drove them on the freeway in our normal way of traveling, that is, in an open car.

I would also point out that it is not only people with known angina pectoris who may suffer from exposure to carbon monoxide. Many individuals who do not have or do not exhibit the typical signs of angina pectoris, the silent angina pectoris, which could do as much damage to them because they do not know about it, and they could get the same effect. Unfortunately, as Dr. Coburn pointed out, we have not studied this. But I do believe that there are some misconceptions about the role that carbon monoxide plays because we tend to consider it only as a stable element in our environment and not as a markedly fluctuating element.

DR. LAWRENCE HINKLE (Cornell University Medical College): For 50 years my concern has been primarily with people who have coronary heart disease, and I have followed more than 1,000 men with coronary heart disease as they go about their work and their daily activities with cardiac recording and so forth. I would be very interested to know what sort of an adverse effect Dr. Coburn thinks is created in an angina patient when he exercises to the point of discomfort and stops. Second, given the fact that

many people who smoke two packs of cigarettes a day or more carry around with them 4 to 7% carboxyhemoglobin all day, what is the relative contribution of the amount of time these people spend near heavily traveled roads?

Third, when we consider that in experimental kitchens running the gas oven will create 50 parts per million in which a housewife may be all day or the better part of the day or that the combination of passive smoking and a poor exhaust fan in a restaurant can create a 35-parts-per-million level very easily for the patrons, where do we stand with 2.7, 2.8 parts per million occurring occasionally?

DR. COBURN: First, what is the adverse effect in patients with angina pectoris if they get pain? I think everybody thinks that this is due to oxygen lack. It is an index of oxygen lack.

The second question had to do with cigarette smokers. Cigarette smokers die 10 to 20 years sooner than nonsmokers. Why they do that could be related to carbon monoxide.

In response to the third question, I thought that illuminating gas in America contains rather a small amount of carbon monoxide. I do not think there is enough in that little burst that comes out before the stove lights to be significant. If it is, I would like to know about it.

DR. HINKLE: I quote experimental studies which I will talk about later.

DR. HERBERT SCHIMMEL (Albert Einstein College of Medicine): You said that cigarette smokers die 10 to 20 years younger. Is that, do you think, a correct figure?

DR. COBURN: I am not an expert on this but as I recall the Summit studies, with two to three packs one dies in one's 50s instead of one's 70s. When one gets into the controversial subject of cigarette smoking, it tends to blur the carbon monoxide story. I am not an epidemiologist who knows the literature well on cigarette smoking. I am very impressed, though, by a lot of it, and I think it is pretty bad.

DR. EISENBUD: Whatever it is, it is too much and avoidable.

DR. SCHIMMEL: Of course, we know that the risk is enormously greater if one smokes. For heart attacks, I believe it is three to one if you are a heavy smoker. And yet, strangely enough, if one looks at the figures, one gets surprised. It is not 10 to 20 years. I think it is much less. I am not sure, but people bandy these numbers about because somehow or other their perception of the way things happen is frequently wrong when one goes into the numbers game; it is not easily translated.

Dr. Hinkle, I think, was on to something very interesting. If a person

gets an angina pectoris pain, the thing for him to do is to stop exercising, and it seems to me that if he has regular angina pectoris pain, he probably will not be exercising very often.

We are in a difficult situation in all this regulation. For whom are we regulating, and are people making their own lifestyle adjustment? Although I smoke, from the public health view I think the smartest thing to do would probably be to compel smokers to grow their own tobacco. They would smoke very little, and it would be much more important than everything else that is being done through regulation.

MISS CAROL WILKINSON (Cornell University Medical Center): Do you think that carbon monoxide plays a significant role in accelerating atherosclerosis?

DR. COBURN: This was an area of research, and it looked like it might be a factor. However, the evidence now supports this view less and less. Some of the earlier work was not reproduced. Some of the experimental work was done on rabbits. So I do not think that this is such strong evidence about carbon monoxide toxicity.

DR. HORVATH: I would add one thing to what Dr. Coburn has said. If one has an anginal attack, I consider that an insult to the arteries, regardless of what effect we immediately assign to it. It does not matter that the victim stops exercising. The organism has been insulted. The organism has to respond to repeated insults, and we know fairly well that any organism subject to repeated insult is unable to survive in this particular environment as effectively as if it did not have the insults. It is important to keep in mind that although we cannot identify the exact part of the myocardium that is destroyed, we can say that if it is insulted sufficiently and frequently, something is going to happen.

There is another question still unanswered, and that has to do with whether we do become adapted to carbon monoxide over a long period of time. Whether this helps the smoker, I do not know.

DR. EISENBUD: I wish more were known about the exposure of carbon monoxide in pre-World War II and immediately post-World War II Japan, where the use of hibachis produced very high concentrations, so high that Americans found it very uncomfortable to spend a night in Japanese-style hotels. I had heard that the Japanese were adapted to higher carboxyhemoglobin. I do not know whether that is so.

DR. HORVATH: We have that information from the Swedish studies. There were many studies on this in Sweden in the early 1920s and 1930s. Nobody refers to them because they are too lazy to read the old literature.